Amendments to the Claims:

Please cancel Claims 17 and 32-34 without prejudice or disclaimer, and amend Claims 1, 7, 9, 18, 22, 24, 31 and 37-38 as set forth below.

1. (Currently amended) A method for enhancing production in a subject of a functional protein from a gene disrupted by the presence of a premature stop codon in the coding region of the gene, comprising administering to the subject an amount of an agent effective to suppress the premature stop codon and an amount of an agent effective to increase transcription of the gene, wherein the agent that is effective to increase transcription of the gene is a fluorinated quinolone or thioguanine, and wherein the agent that suppresses the premature stop codon is administered at a dose lower than the dose that would be required to produce the same amount of functional protein in the absence of the agent that increases transcription.

2. (Original) The method of claim 1, wherein the agent that suppresses the premature stop codon is an aminoglycoside antibiotic.

3. (Original) The method of claim 2, wherein the aminoglycoside antibiotic is selected from the group consisting of gentamicin, geneticin, paromomycin, hygromycin, G-418, kanamycin, amikacin and tobramycin.

4. (Original) The method of claim 3, wherein the aminoglycoside antibiotic is gentamicin.

5. (Withdrawn) The method of claim 3, wherein the aminoglycoside antibiotic is geneticin.

6. (Original) The method of claim 1, wherein the agent that increases transcription of the gene is an agent that activates a promoter of the gene.

7. (Withdrawn and Currently amended) The method of claim 1 [[6]], wherein the agent that is effective to increase transcription of the gene ~~activates a promoter of the gene~~ is a fluorinated quinolone.

8. (Withdrawn) The method of claim 7, wherein the fluorinated quinolone is ofloxacin.

9. (Currently amended) The method of claim 1 [[6]], wherein the agent that is effective to increase transcription of the gene ~~activates a promoter of the gene~~ is thioguanine.

10. (Original) The method of claim 1, wherein the production of functional protein is enhanced by a factor of at least 7-fold relative to an untreated control.

11. (Original) The method of claim 10, wherein the production of functional protein is enhanced by a factor of at least 10-fold relative to an untreated control.

12. (Original) The method of claim 11, wherein the production of functional protein is enhanced by a factor of at least 20-fold relative to an untreated control.

13. (Original) The method of claim 12, wherein the production of functional protein is enhanced by a factor of at least 30-fold relative to an untreated control.

14. (Original) The method of claim 1, wherein the production of functional protein is enhanced by a factor of at least 2-fold relative to the production obtained using only the agent that suppresses the premature stop codon.

15. (Original) The method of claim 14, wherein the production of functional protein is enhanced by a factor of at least 3-fold relative to the production obtained using only the agent that suppresses the premature stop codon.

16. (Original) The method of claim 1, wherein the production of functional protein is enhanced to a level that corresponds to at least 10% of the level of functional protein generated from a corresponding native gene in which a premature stop codon is absent.

17. (Canceled)

18. (Currently amended) The method of claim 1[[7]], wherein the lower dose of the agent that suppresses the premature stop codon results in decreased toxicity.

19. (Original) The method of claim 1, wherein the disruption of the gene is associated with a genetic disorder.

20. (Original) The method of claim 19, wherein the genetic disorder is selected from the group consisting of thalassemia, hemophilia A, hemophilia B, von Willebrand's

disease, a p53 related cancer or disorder, a colorectal cancer, cystinosis, cystic fibrosis, Duchenne muscular dystrophy, Tay-Sachs disease, Wilms tumor, retinoblastoma, neurofibromatosis, ataxia telangiectasia, Hurler's syndrome, mucopolysaccharidosis I, and late infantile neuronal ceroid lipofuscinosis.

21. (Original) The method of claim 20, wherein the genetic disorder is ataxia telangiectasia.

22. (Withdrawn and Currently amended) The method of claim 21, wherein the agent that is effective to increase transcription of the gene ~~activates a promoter of the gene~~ is a fluorinated quinolone.

23. (Withdrawn) The method of claim 22, wherein the fluorinated quinolone is ofloxacin.

24. (Currently amended) The method of claim 21, wherein the agent that is effective to increase transcription of the gene ~~activates a promoter of the gene~~ is thioguanine.

25. (Original) The method of claim 21, wherein the agent that suppresses the premature stop codon is gentamicin.

26. (Withdrawn) The method of claim 21, wherein the agent that suppresses the premature stop codon is geneticin.

27. (Original) The method of claim 19, wherein the genetic disorder is treated.

28. (Original) The method of claim 1, wherein the gene is a tumor suppressor gene.

29. (Original) The method of claim 28, wherein the tumor suppressor gene is BRCA1, BRCA2, PTEN, NF1, NF2, MLH1, MLH2, VHL, WT1, TSC1, TSC2, and/or ATM.

30. (Original) The method of claim 28, wherein the enhanced production of the functional protein is effective to treat a tumor in the subject.

31. (Currently amended) A method for enhancing production in a subject of a functional protein from a gene, where production of the protein is disrupted by an ~~genetic~~ exon skipping mutation, comprising administering to the subject an amount of an agent effective to suppress the ~~genetic~~ exon skipping mutation and/or correct a defect caused by the exon skipping mutation, and an amount of an agent effective to increase transcription of the gene, wherein the agent that suppresses the exon skipping mutation and/or corrects a defect caused by the exon skipping mutation is a 2′-*O*-methyl phosphorothioate oligonucleotide combining an antisense sequence and an exonic splicing enhancer sequence, sodium butyrate, or aclarubicin, and wherein the agent that is effective to increase transcription of the gene is a fluorinated quinolone or thioguanine.

32-34. (Canceled)

35. (Original) The method of claim 31, wherein the agent that increases transcription of the gene is an agent that activates a promoter of the gene.

36. (Original) The method of claim 31, wherein the genetic mutation is associated with a genetic disorder.

37. (Withdrawn and Currently amended) The method of claim 31 ~~33~~, wherein the mutation is associated with spinal muscular atrophy (SMA).

38. (Currently amended) The method of claim 36 ~~31~~, wherein the genetic disorder is treated.